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Catatonia Yesterday, Today, Tomorrow

Amresh Srivastava

University of Western Ontario, amresh.srivastava@sjhc.london.on.ca

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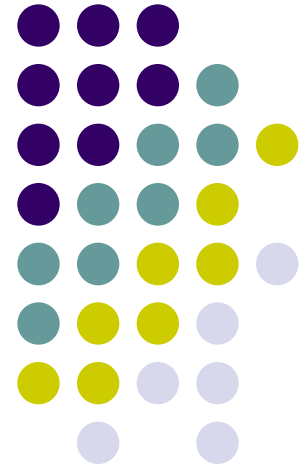
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Catatonia

Yesterday, today, tomorrow

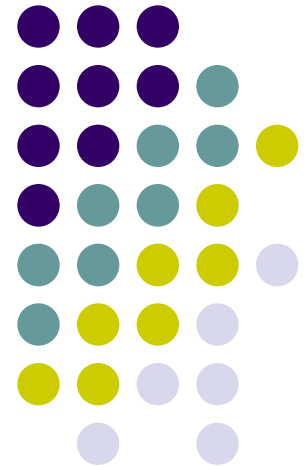
Amresh Shrivastava
Assistant Professor
University of western Ontario, London, Canada
&
Physician Team leader,
Early psychosis Programme,
RMHC- St.Thomas
E-mail: asrivas9@uwo.ca



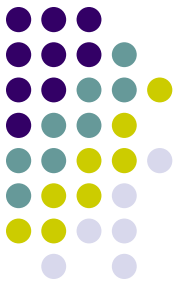
Disappearance vs.. re-emergence

- Challenging,
- High mortality
- Life-threatening
- Beyond psychiatry
- Offers interface with medicine and neurology
- Constantly evolving in understanding

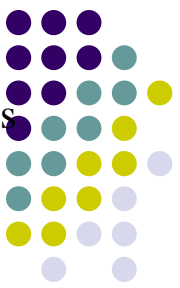
52 publications in 2006



Catatonia: Case vintage.



- A 40-yr.old man treated for psychosis with antipsychotic and antimanic
- medications since age of 16, was attending a community clinic, and stabilized
- with lithiumand chlorpromazine therapy.
- Encouraged by stability of condition therapist prescribed olazapine.
- Within a few weeks patient again became psychotic. Perphanizine was prescribed, within a day he became febrile, mute and rigid. Hospitalized and diagnosed as NMS, transferred to tertiary care facility
- APD was discontinued, further treated with large doses of bromochriptine & dantrolene. Repetitive movements prompted diagnosis of epilepsy, so anticonvulsants were administered.
- Lorazepam prescribed in low dose controlled infrequent agitation but he remained mute and rigid, required total nursing care
- After few weeks he was unable to stand; hands and legs were in rigid, immobile posture.
- A gastrostomy was done to permit feeding, he developed pulmonary and bladder infection requiring antibiotics
- He spent four months in intensive medical care and after that a visiting consultant [psychiatrist] recommended lorazepam in high doses.
- When the daily dose was increased to 12mg he responded to commands and smiled at his parents, though he remained mute
- ECT was recommended but the hospital had no facility, so he was transferred
- When his mother was signing the consent for ECT she recalled that he had a similar episode of rigidity mutism and psychosis at 16 years of age and responded to ECT



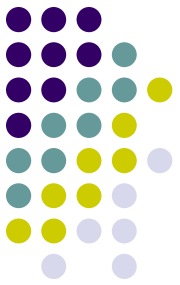
- Lorazepam was reduced to 6 mgs per day and bilateral ECT begun
- After 4 treatments he recognized his parents, vocalized, smiled, was less rigid and took oral feedings
- By 9th treatment he was verbally responsive but 4 months of rigidity & forced bed rest had left him with limb contractures and such badly impaired movements that he was unable to stand or use his hands to feed himself.
- Both catatonia and psychosis were relived
- After 22 ECT he was transferred to rehabilitation center and 4 months later he was again able to walk, use his hands and care for himself.

➤ COMMENTS

- This patient's ordeal was prolonged by several clinical missteps. Persisting in a failed trial with bromocriptine and dantrolene was not correct
- Generally if an acutely ill patient has not improved substantially within 7-10 days of treatment, that treatment needs to be reconsidered and probably changed
- Catatonic features interfering with general medical health, even catatonic features attributed to antipsychotic drugs improve substantially within several days when properly treated
- For this patient inadequate nursing care allowed contractures to develop
- The unavailability of ECT at a tertiary care hospital was indefensible.
- A long ill catatonic patient with joint contractures was successfully treated with Acts.

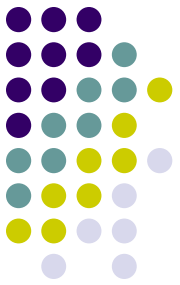
➤ **REFERENCE:**

- *Mashimo et al 1995 quoted in Catatonia by Fink and Taylor 2003 Cambridge University press; pp 21.*



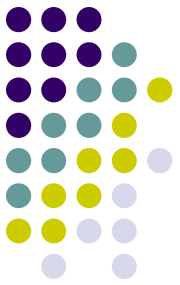
Catatonia is one clinical condition
which reminds us that
patients do not follow books but
books are written by following patients

Catatonia: A life threatening condition.

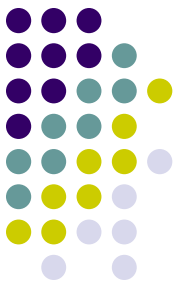


- ✓ A syndrome
- ✓ Catatonia is currently considered to be a psychomotor syndrome
- ✓ Remarkable constellation of motor and behavioral signs and symptoms that often occurs in relation to neuromedical insults.
- ✓ Structural brain lesions, intrinsic brain disease, and other systemic disorders that affect the brain, as well as
- ✓ 'idiopathic' psychiatric disorders have been found to be associated

Catatonia: Historical evolution



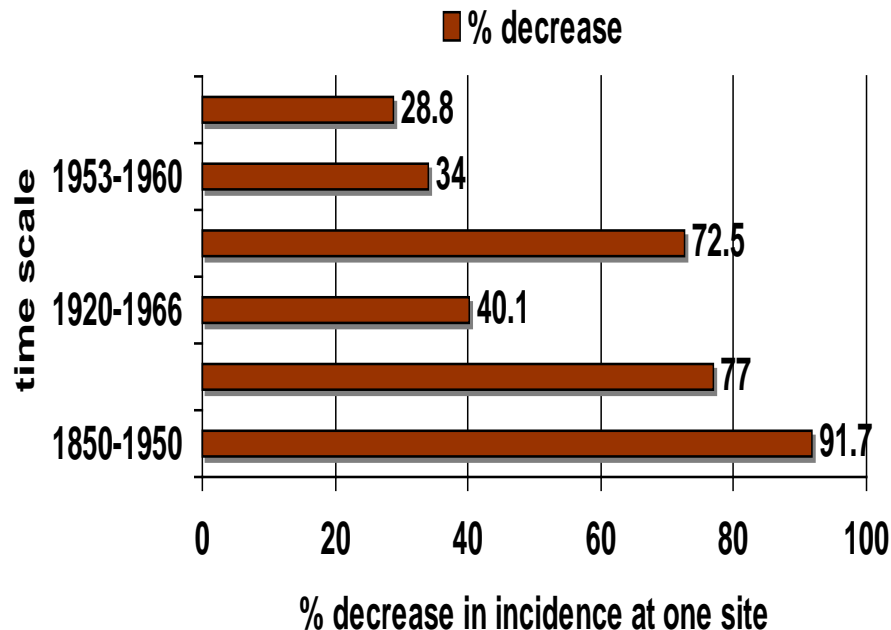
- Catatonia or tension Insanity- Kahibaum 1874
- Catatonia-dementia praecox kraepelin 1899
- Symptoms as manifestation of Freudian complexes-Bleuer 1911
- With MDP - kahlbaum 1874
- Motility Psychosis-Wenicke 1900
- Cycloid Motility psychosis- Kleist 1912 and leonhard 1957
- Catatonic schizophrenia-a group of Cerebral System Disorder - Kleist 1923
- In Childhood & adolescence- Raecke 1909
- Catatonia & Hysteria-Charcot 1886
- Catatonia & OCD-Bleuler 1911, Kruger 2000
- It was also well known that it could occur secondary to neurological, toxic-metabolic and infectious etiologies.
- Identified exclusively with schizophrenia as late as DSM III/ III-R
- This limitation changed in DSM-IV.



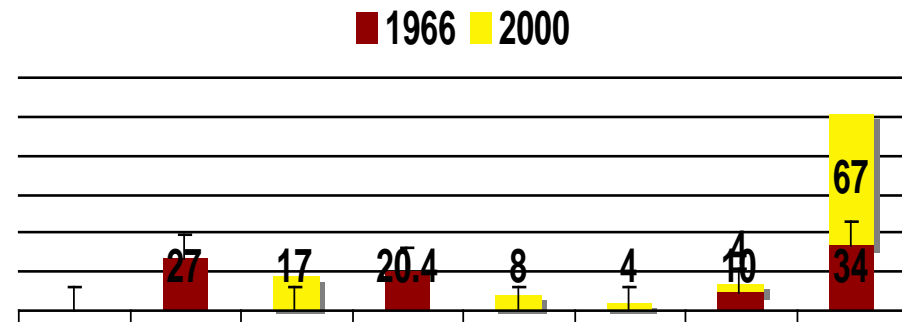
Epidemiology (definition & concept)

1. rates of catatonia- a) General psychiatric Condition-7-175 hospitalized acute psychosis b) mood disorder- 13-31% c) schizophrenia- 6% in 1850 & 0.5% in 1950

incidence of catatonia

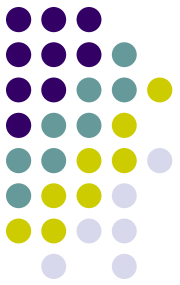


change in diagnosis

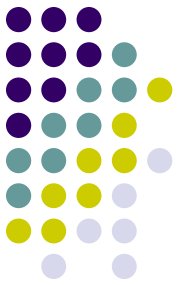


diagnosis

Nosology



- Motility psychosis
- Catatonic schizophrenias
- A- unsystemetic catatonic,
schiz, periodic catatonia
- B-systematic- schizophrenias
- 1.Para kinetic
- 2.mannerist
- Pros kinetic
- Negativistic
- Speech prompt
- Speech inactive
- Nosologic validity continues to be debated
- Concept & descriptive psychopathology-still insufficient
- Study of catatonia can be advanced by--
- Adopt the approach of 'catatonia across psychiatric illness'
- Reject notion of subtype or modifier
- Postulate as ' separate entity'



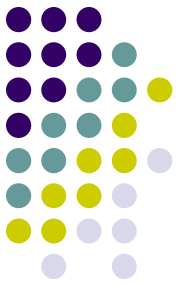
Current phenomenological classification

As per DSM-IV; Now it is categorized as

- Catatonic disorder due to general medical condition
- Mood disorder with catatonic features
- The catatonic type of schizophrenia.

Historically , Pre and post neuroleptic era has been found strongly associated with incidence of catatonia.

- Psychogenic catatonia
- Neuroleptic induced catatonia -NMS
- [catatonia preceding NL administration and developing into NMS]
- Are possibly most suitable clinical classification.



Catatonia in psychiatric classification: the evidence

- Catatonia is a condition qualifying for finding its own place in various diagnostic systems. Because:-
- Catatonia is common
- Is Identified as a syndrome
- Can be Delineation from other syndromes[differential diagnosis]
- Catatonia is also known by by other names
- Known to have Good response to specific treatment

Common causes of catatonia

- Mood disorder
- General medical and neurological conditions
- Non-affective psychosis
- Genetic form of catatonia

Proposed category for classification

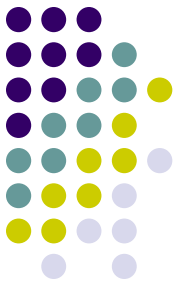
Max Fink & Taylor; 2003



- DSM Code xxx.0
- Catatonia
- Code.xxx.1 Nonmalignant catatonia
- Code.xxx.2 delirious catatonia
- Code.xxx.3 malignant catatonia, NMS, serotonin syndrome
- Specifier
- Code.xxx.x1 secondary to mood disorder
- Code.xxx.x2 secondary to gen.med.condition
- Code.xxx.x3 secondary to neurological dis.
- Code.xxx.x4 secondary to psychiatric disorder

Proposed diagnostic criteria

Max Fink & Taylor; 2003



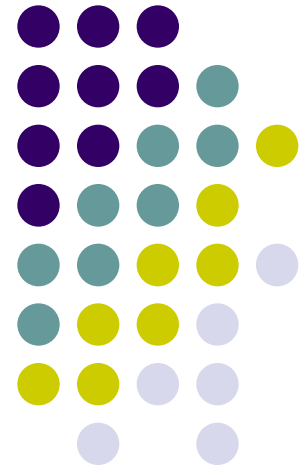
- **A.**immobility, Mutism, or stupor of at least 1 hours duration, associated with at least one of the followings: catalepsy, autonomic obedience, or posturing, observed or elicited, on two or more occasions.
- **B.**in absence of immobility, Mutism or stupor, at least two of the followings: stereotypy, echophenomena, catalepsy, autonomic obedience, posturing, negativism, gagenthalten, ambitendency

Catatonia & NMS {NL induced catatonia},
a lethal and malignant syndrome
with high mortality-NMS : first description 1960
by Delay et al.

**Share a common pathophysiology,& clinical
manifestation**

**While most cases of catatonia do not meet
criteria for NMS , all unequivocal cases of NMS
appear to meet criteria for catatonia.**

**Though the syndrome has been described in different
times , there is remarkable amount of similarity**



The classical features of catatonia:

about 42 symptoms have been identified

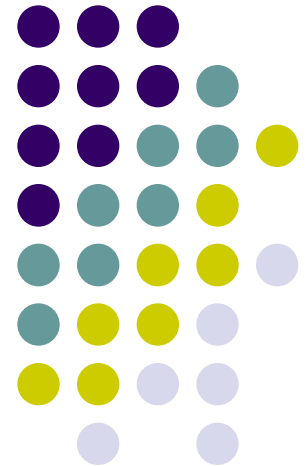
Catatonia has three groups of symptoms:

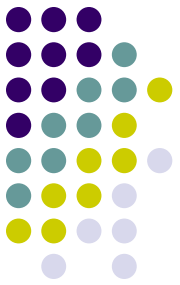
- motor symptoms
- behavioral symptoms
- emotional symptoms

All three have different underlying pathophysiological mechanism

Mutism
Immobility
Staring

Posturing
Negativism
Withdrawal





Subtypes and classification:

Main subgroups are:

Malignant and Non-malignant Catatonia

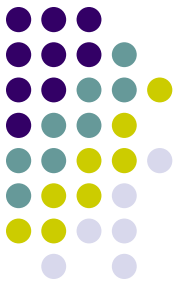
- ✓ Catatonic withdrawal
- ✓ Catatonic excitement
- ✓ Alternating
- ✓ Periodic [excitement alternating with stuporous state]
- ✓ Lethal catatonia
- ✓ Catatonic stupor
- ✓ Malignant
- ✓ Simple - non-malignant
- ✓ Pernicious
- ✓ NMS
- ✓ variants

➤ Clinical description, course and outcome

Catatonia :

*Idiopathic:*___without brain atrophy & -with brain atrophy

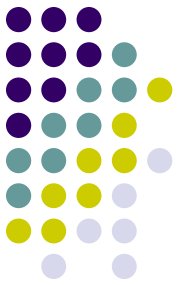
Associated with bipolar disorder; major depression other affective disorder: schizophrenia; other psychiatric disorder
[Psychogenic]



Catatonia secondary to:(Medical Catatonia)

- TLE
- other Seizure Disorder
- brain tumor
- brain trauma
- encephalitis- post encephalitis state
- cerebro vascular diseases
- focal brain lesions
- akinetic mutism
- Parkinson's
- Toxic encephalopathy
- Metabolic encephalopathy
- Other medical disorders
- NMS
- Neuroleptics
- Other prescribed psychotropics
- Illicit psychotropics

Phencyclidine exposure, SLE, Corticosteroids; disulfiram Porphyrria



Assessment of catatonia

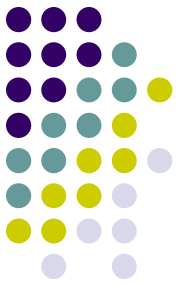
by Braunig catatonia rating scale

The scale contains 21 items

(16-motor symptoms & 5 behavioral symptoms rated from 0-4)

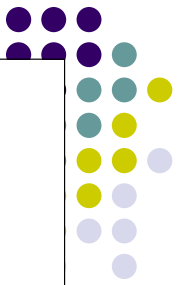
- Motor excitement
- Verbigeration
- Stereotypis
- Motor inhibition
- Iteration
- Grouping
- Impulsivity
- Mutism
- Grimacing
- Exaggerated responsiveness
- blinking
- Mannerism
- Jerky movements
- Genhalten
- Rigidity
- Negativism
- Parakinesis
- Postural
- Waxy flexibility
- Rituals
- Automatic obedience

Mood disorder vs. schizophrenia

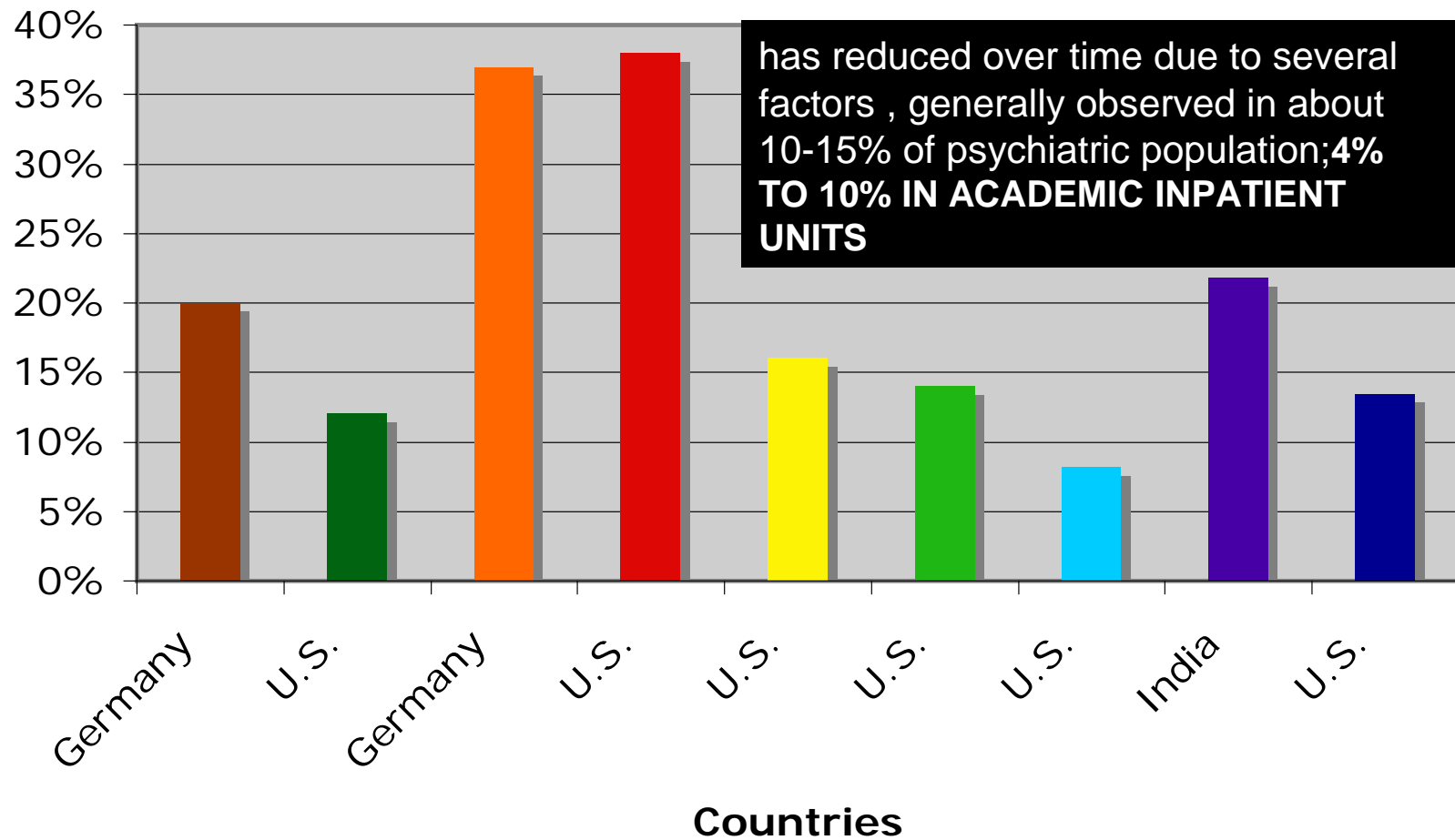


Catatonic spectrum disorders: depending upon shared neurobiology

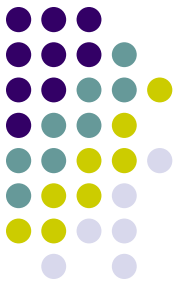
- Lethal malignant catatonia - high mortality
- NL induced catatonia, NMS,
- Serotonin syndrome
- Classical catatonic stuporous state
- Various independent catatonic symptoms
- Catatonia variants
- Non-malignant simple catatonia
- Autism spectrum disorder



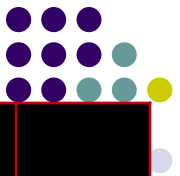
Incidence of catatonic schizophrenia in various countries



Are we witnessing the disappearance of catatonic schizophrenia? Symptoms or syndrome.??



• E.Bleuer	1991	>33	▪ Huber	1979	4.7
• Kraepelin	1913	19.5	M.Bleuer	1972	33.6
• Mayor-Gross	1932	30	Muller	1976	21.5
Leonhard	1938-68	35.4	Warthen	1967	7.5
Leonhard	1969-86	30	Morrison	1973	10
Astrup	1962	25.6	Kasper	1975	5
fish	1964	30.5	Strass	1981	5.7
Huber	1957	27	Kane	1988	2
			Stieglitz	1992	8



	Catatonia in depression	
feature	motoric immobility stupor;extreme agitation;extreme negativism;posturing;stereotyped movements,mannerisms,or grimacing and echolalia or echopraxia	
treatment	BZ with continued oral administration, When , no relief, urgent provision for ECT After catatonic symptoms are relieved Tx should continue with ADD,Lithium, APD or combination	
outcome	Efficacy of ECT usually appears after few treatments, ECT may initially be administered daily	

Catatonia in mania

features

develop in 1/3 of patients.

are--motor excitement, Mutism, and stereotypic movements, patients exhibiting stupor may go on to show more typical signs of mania, greater episode severity, mixed states, and poorer short-term outcome,

treatment

Lorazepam is effective,

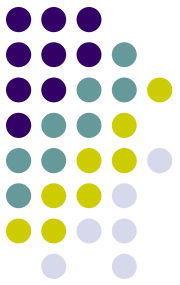
ECT is most effective , regardless of etiology, should be considered if BZ fails.

outcome

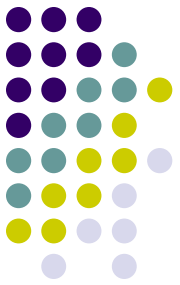
NL generally have exhibited poor efficacy,

Leonhard: 2 types of MDP with catatonic features; with or without catatonia; significantly differing on mean number of days in hospital

High risk or At-risk or Vulnerable for catatonia & poor prognosis

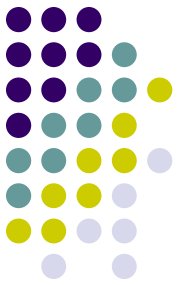


- Male gender
- Adolescent onset
- Presence of autistic traits
- Obstetric events
- Post-natal brain insult
- Soft neurological signs
- NL sensitivity
- Family history of catatonia
- Catatonia generally has good outcome but presence of catatonic symptom has poor prognosis of schizophrenia or mania



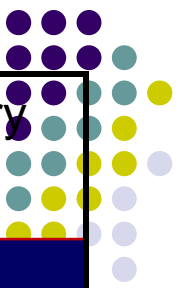
NMS & its Risk factors

- Mortality has decreased from 20% to 10% and incidence has also decreased.
- Life time incidence - about 0.2% amongst antipsychotic users
- Hyperthermia and rhabdomyolysis may lead to renal failure
- CPK rises dramatically [even up to 60K units]
- **Risk factors include:-**
- Rapid dose escalation of HP , first generation APD
- Parenteral administration of APD
- Underlying neurological impairment
- NMS is probably less common with second generation APD than with first one.
- Incidence with first generation is low now possibly because of low dosage than in the past.
- Polypharmacy, concomitant anticholinergics, Lithium

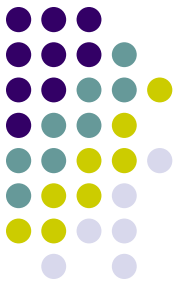


NMS & its Risk factors

- May be fatal, if untreated,
- Tx. Includes : temperature control by cooling, fluid correction, DA agonist-Bromocriptine 2.5 mg QDS, muscle relaxant, dantrolene 1-2.5 mg/kg, IV 6 hourly
- ECT is indicated if catatonia related to NMS persists or response is inadequate with drugs,
- Need for APD should be carefully assessed, before resumption,
- When another trial of APD is attempted, second generation , particularly clozapine are preferred,
- A re-challenge should begin with low dose and slow titration



NMS variables	Death N=17	Sequelae N=10	Full recovery N=22
age	50.3	51.3	43.1
female	59%	60%	23%
Psychomotor excitement	41%	60%	36%
dehydration	54%	80%	64%
Lithium	18%	30%	9%
Antiparkinsons	76%	30%	77%
I/M NL	35%	30%	14%
Dose NL/day	789	486	728
Loading dose NL/d ²	121	51	127
dantrolene	59%	80%	59%
bromcriptine	14%	30%	41%
Max CPK 1000 IU/L	13.9	25.5	49.2
Max body temperature	39.4	40	38.6

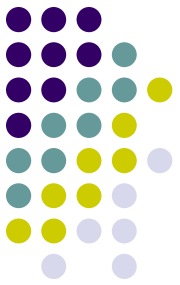


Differential Diagnosis

organic, psychogenic, drug induced, idiopathic,

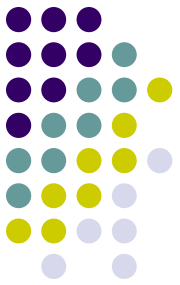
- Idiopathic primary catatonia
- Secondary catatonia
- Lethal catatonia
- Catatonic excitement

- Diagnosis is essentially clinical supported by
- measurement and rating scales,
- Rule out other causes
- presents as emergency ,
- move very quickly and systematically
- Often diagnosing catatonia in schizophrenia vs. mania is difficult at the beginning.



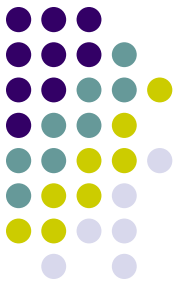
Clinical dilemmas and challenges

- Very severe cases has less problem,
- Most of the difficulties arise in **borderline cases**.
- **New contact** : most would adopt 'rule out' .
- Time length for definitive Tx is crucial
- Severe excitement is a management problem
- Partial syndromes only
- **When diagnostic criteria is not fulfilled**
- Presence of high fever and h/o NL
- Rapid cycling and periodic catatonia
- When response does not progressed



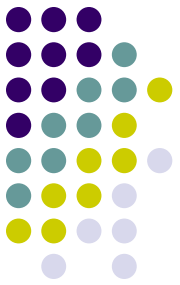
Clinical dilemmas and challenges

- When response does not sustain
- Frequent relapses
- Determining psychiatric diagnosis schizophrenia vs. mood disorder
- Presence of psychosis;
- Use Antipsychotic ; to be or not to be
- Should CSF be checked / routinely
- Deciding about ECT in medically compromised
- Therapeutic or service set up
- Issues of transfer
- Issues around consent and capacity



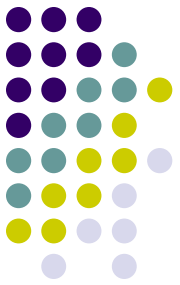
Clinical dilemmas and challenges

- Late onset, paediatric, in pregnancy
- Planning relapse prevention
- Which antipsychotic in catatonic Schizophrenia after termination with ECT and which MS and ADD in Bipolar Disorder?
- Virtually all APD typical or atypical have been known to cause NMS, Incipient NMS or its variants, including clozapine.
- All atypical APD have shown some degree of response in NMS and catatonia?
- Clinical guidelines are not clear and experience is the best guide.



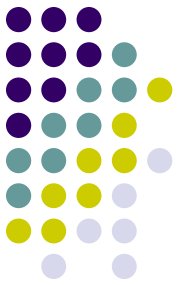
APA practice Guidelines 2006.

- “there are at present no treatment strategies specific to the various subtypes [of schizophrenia] with exception to the use of BZ for Catatonia”
- In Mania use BZ and ECT
- In depression; ‘concurrent ADD use’ and BZ with ECT, “after catatonic features are relieved TX may be continued with ADD,Lithium,APD or a combination..”



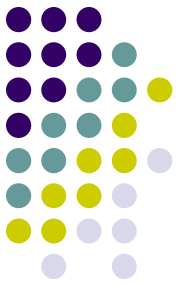
Special conditions

- Catatonia-autism spectrum
- Catatonia in childhood and adolescence
- Late-onset catatonia
- Catatonia in liver transplant
- Post-partum catatonia
- Catatonia in co-morbid medical condition like hypertension

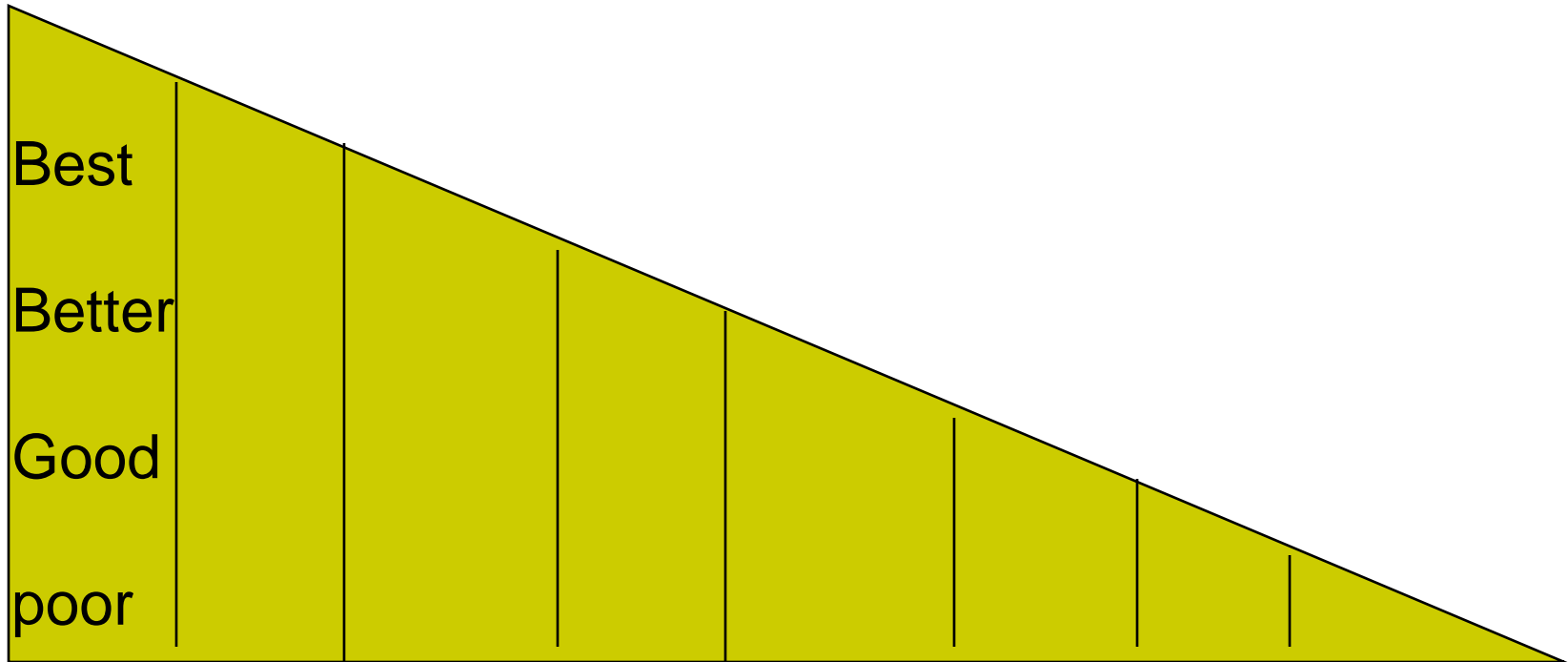


Clinical advances

- Catatonia is an infrequent but severe condition in young people, and is usually associated with schizophrenia.
- Obstetric complications and neurological abnormalities in neuroleptic-naïve psychotic patients.
- Blueprints for the assessment, treatment, and future study of catatonia in autism spectrum disorders.
- Shared susceptibility region on chromosome 15 between autism and catatonia.
- Classification matters for catatonia and autism in children.
- NMS in adolescents after brief exposure to olanzapine
- Catatonia in Alzheimer's Lewy body dementia after Donepezil
- Malignant and late onset catatonia
- Catatonia after single dose of 'ecstasy'



Relative Prognosis



mood
disorder
without
catatonia

depression
With
catatonia

2007

periodic
catatonia

cycloid
psychosis
with
catatonia

as9

Bipolar
disorder
with
catatonia

catatonic
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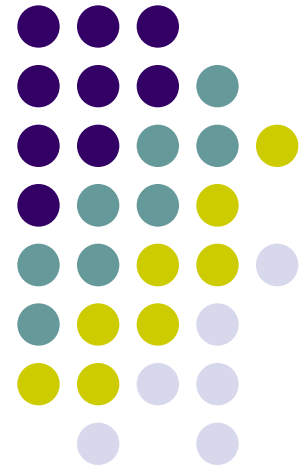
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Fundamental Problem

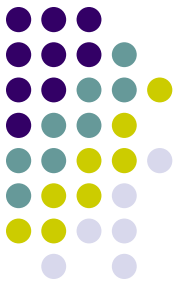
A disorder of Motivation and Movement link

Catatonic patients are well able to
initiate movements, but they are apparently
unable to terminate the movement
once initiated in an appropriate way.

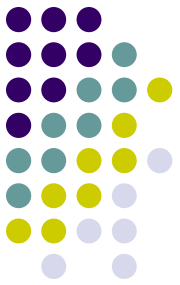
In contrast to initiation, neural network study of
underlying termination of movement has been
neglected in the research to date.



All information put together, it appears that the

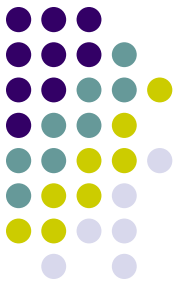


- Motor symptoms ----- nigrostriatal, basal ganglia and motor cortex
- Emotional symptoms ---- prefrontal and limbic cortex
- Behavioral symptoms---mesolimbic cortex
- All three are interdependent because of 'circuits and loops which are closed within these structures.
- That there is a pre-existing brain insult present in vulnerable candidates



Various hypothesis have been proposed.

- Neurotransmitter hypothesis
- Universal field hypothesis
- Vulnerability theory because of brain structural changes
- Emotional-motor activation paradigm
- Motivation-movement paradigm
- Neurochemical: increased CPK and Low serum Iron
- Neuronal Circuits paradigm
- Restitutive dopamine hypothesis
- Top-down modulation”: A Neuropsychiatric hypothesis: parallel from Parkinson's disease

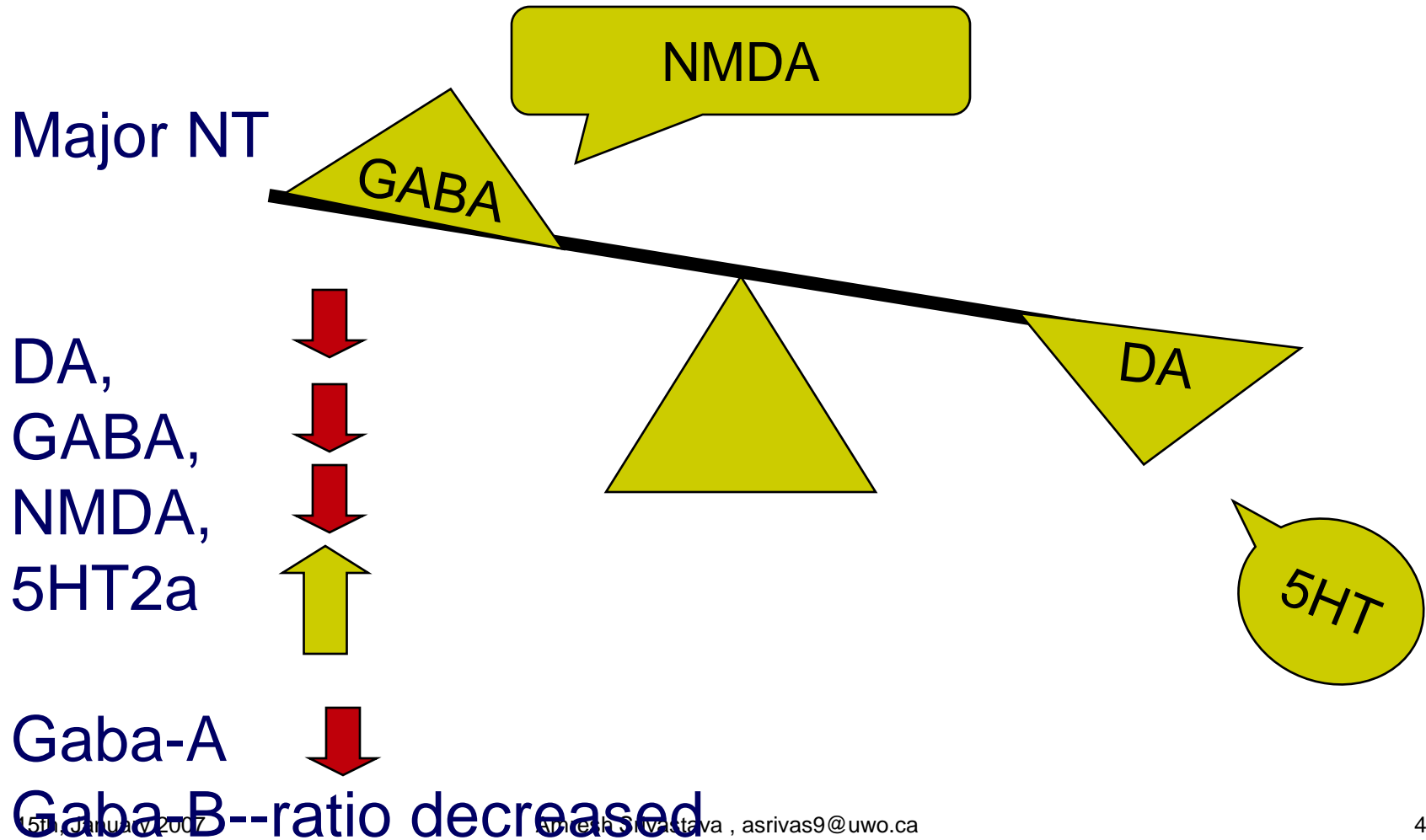
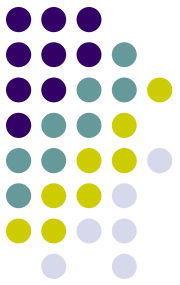


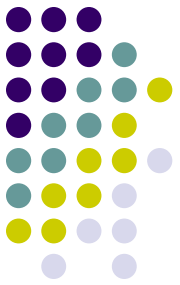
Dopamine hypo-activity

- There is dysfunction in neurotransmission in cortical and sub-cortical areas.
- The dysfunction is a result of a combination of NT involvement.
- Leading NT involved are DA, GABA, 5HT, NMDA
- There is hypofunction D2 activity in various cortical areas mesolimbic, nigrostriatal, motor, prefrontal and orbitofrontal areas
- Decreased D2 function is also found in caudate nucleus, nucleus accumbens, pallidum and thalamus.

This hypothesis proposes that it is the interaction of these systems that predisposes, initiates, and maintains the twin syndromes of catatonia & NMS

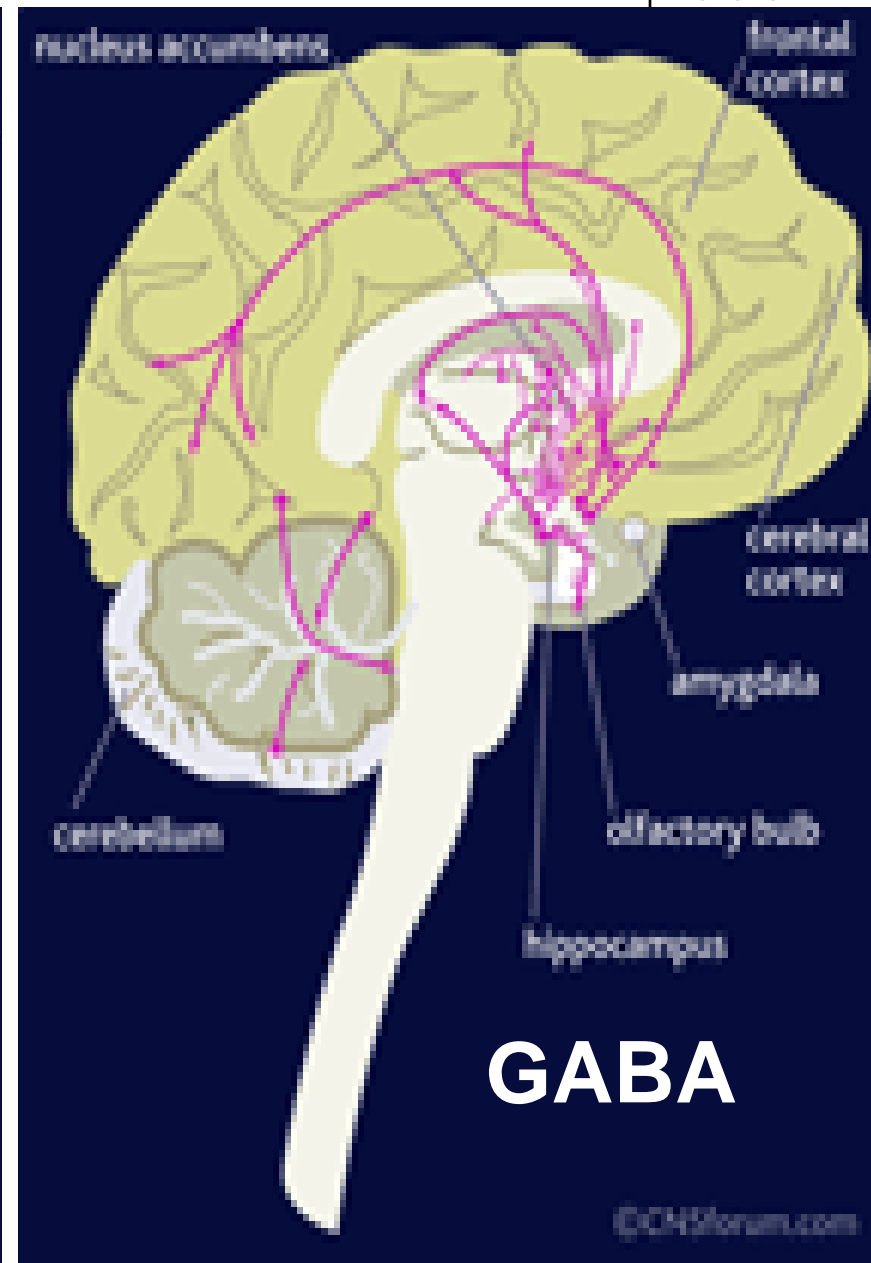
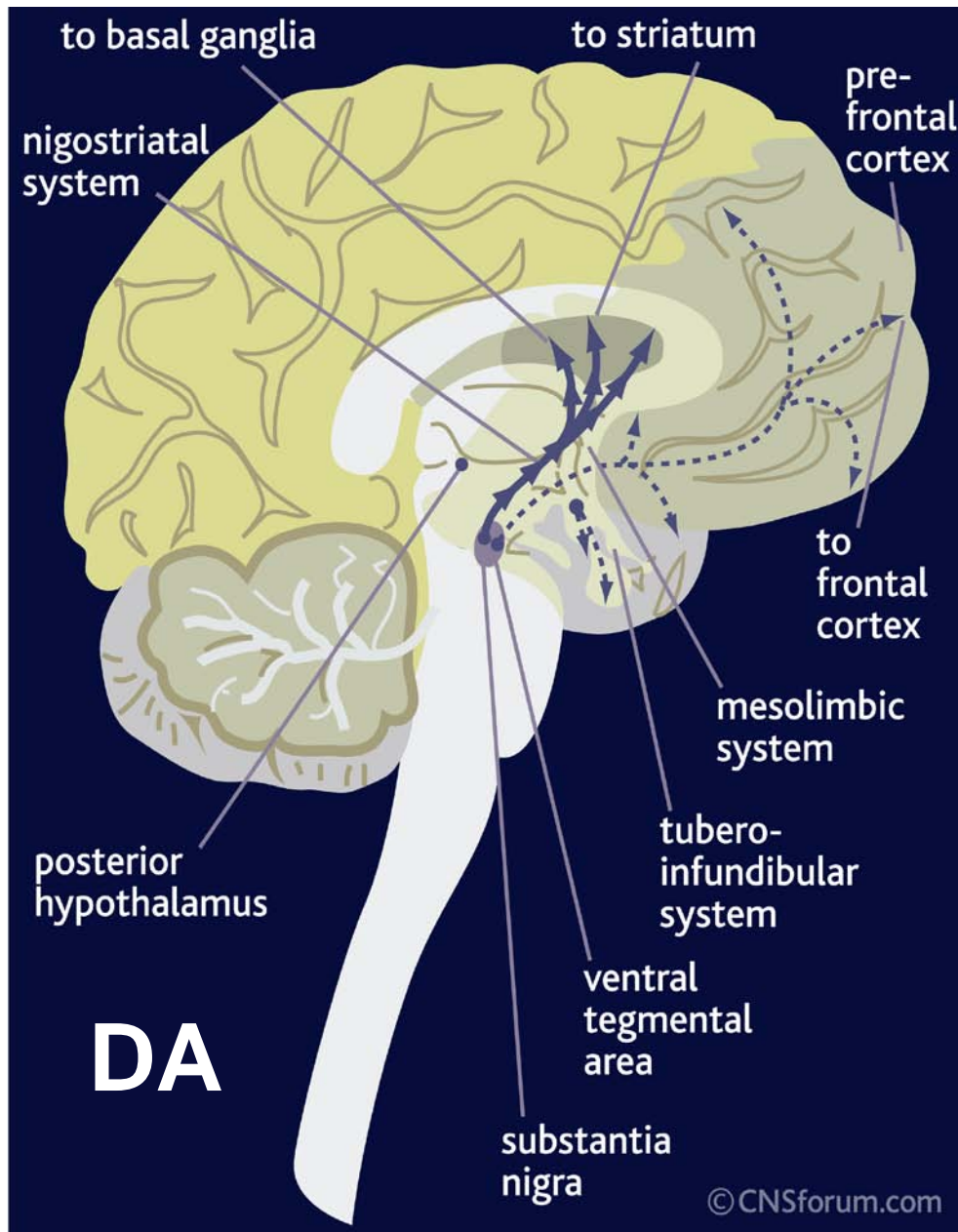
Catatonia is a delicate balance between dopamine and Gaba neurons





Gaba and major brain areas

- The **ratio of GABA A and GABA B** may play a role in development of catatonia. **There is Hypo activity at GABA A receptor**; Lorazepam, a GABA agonist is **effective in catatonia**
- ✚ Serotonin **Hyperactivity** at the 5-HT 1A receptor
- ✚ **hypo activity at 5HT 2A** receptor
- ✚ Glutamate **Hypo function** of NMDA Receptor
- **Major Brain regions implicated in catatonia are:**
- Limbic system - nucleus accumbens
- Thalamus Caudate nucleus Motor area
- Fronto-orbito cortex Right posterior parietal cortex
Mesolimbic and mesostriatal cortex,



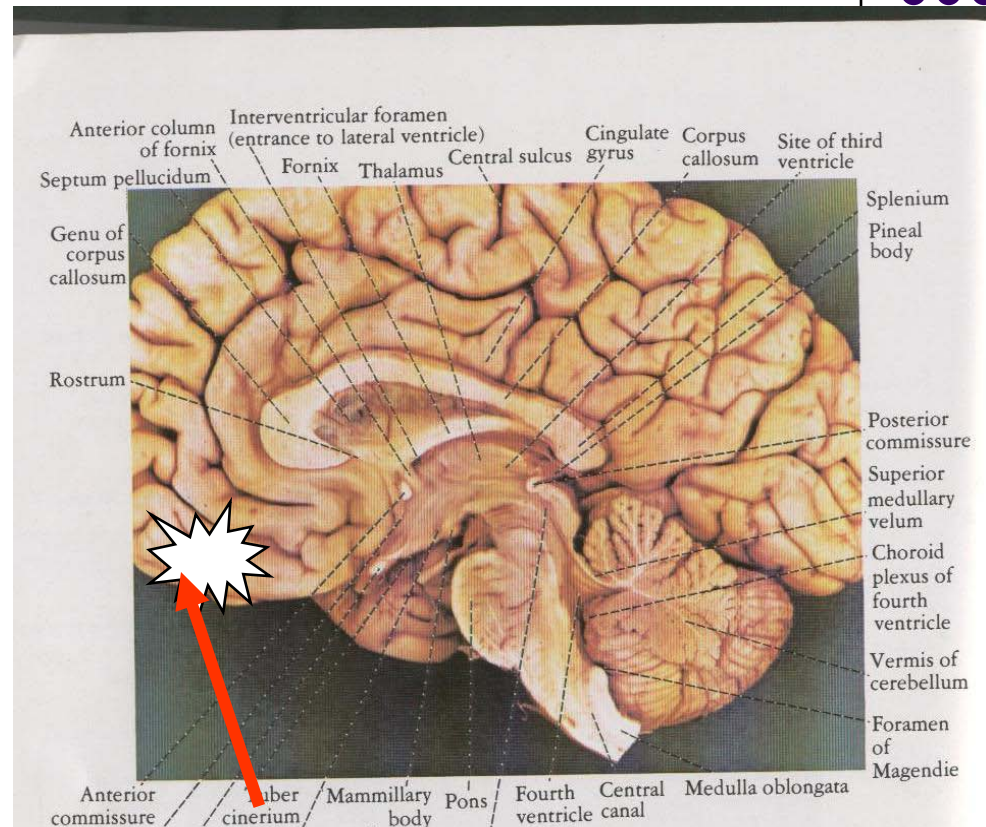


catatonia	Findings of brain imaging
Structural changes Neuropathological findings	Caudate Nucleus:Palladium; nucleus accumbens;Thalamus shows Decreased cell density
Functional changes	<p>reduced activity in medial orbitofrontal cortex during negative emotional stimulation,suggesting possible initiation point.</p> <p>imaging and neuropsychology indicate a relationship between deficits in visual-constructive function & decreased rCBF in the right posterior parietal cortex, alteration in right posterior parietal function may count for the deficit in termination of movements responsible for motor symptoms of posturing.</p>

Movement control

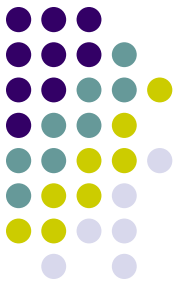
In healthy individuals termination of movement involves
the **right posterior parietal cortex** because the registration.

on line monitoring of the respective **spatial position** of the movement may be of central importance for an appropriate termination.



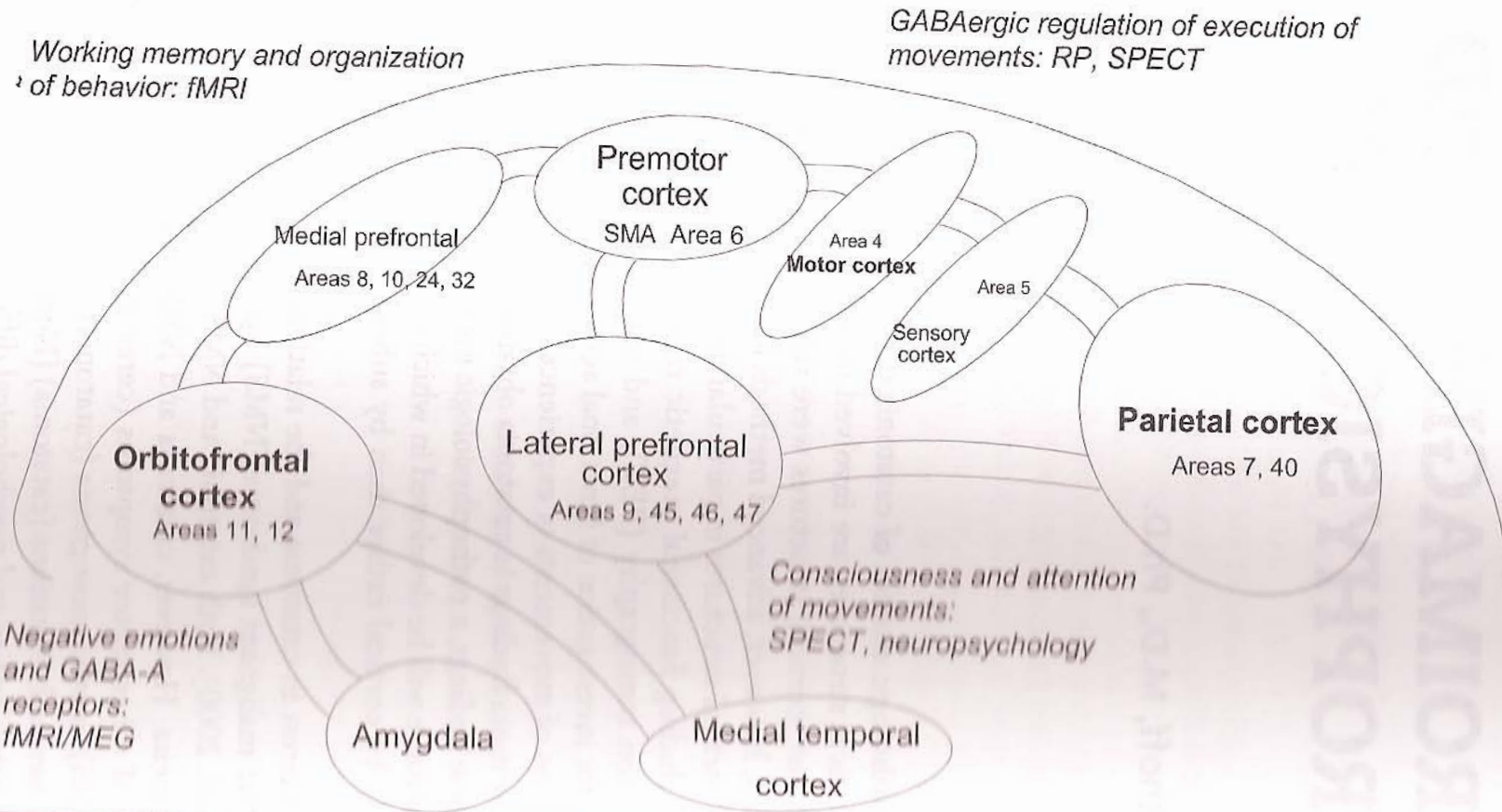
Negative emotional processing in the **right medial orbitofrontal cortex** may be particularly altered in catatonia with an abnormal functional connectivity to the premotor/motor cortex

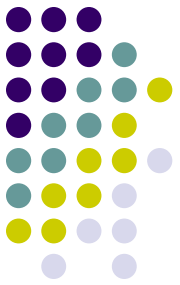
Neuronal circuits and catatonia



Cognitive, behavioral and emotional circuits

- Each circuit has **cortico/limbic, striatal, pallidial/nigral, and thalamic nodal points** with the loops closed by thalamocortical connections.
- Any neuromedical or psychiatric disturbance significant enough to disrupt the GABA-DA balance in the mesostriatal-mesocorticolimbic medial forebrain bundle of DA tracts
- with terminal fields in nucleus accumbens, the anterior cingulate, and the prefrontal cortex system anywhere along the circuitry **will potentially set off a catatonic response.**





Dopamine restitutive hypothesis

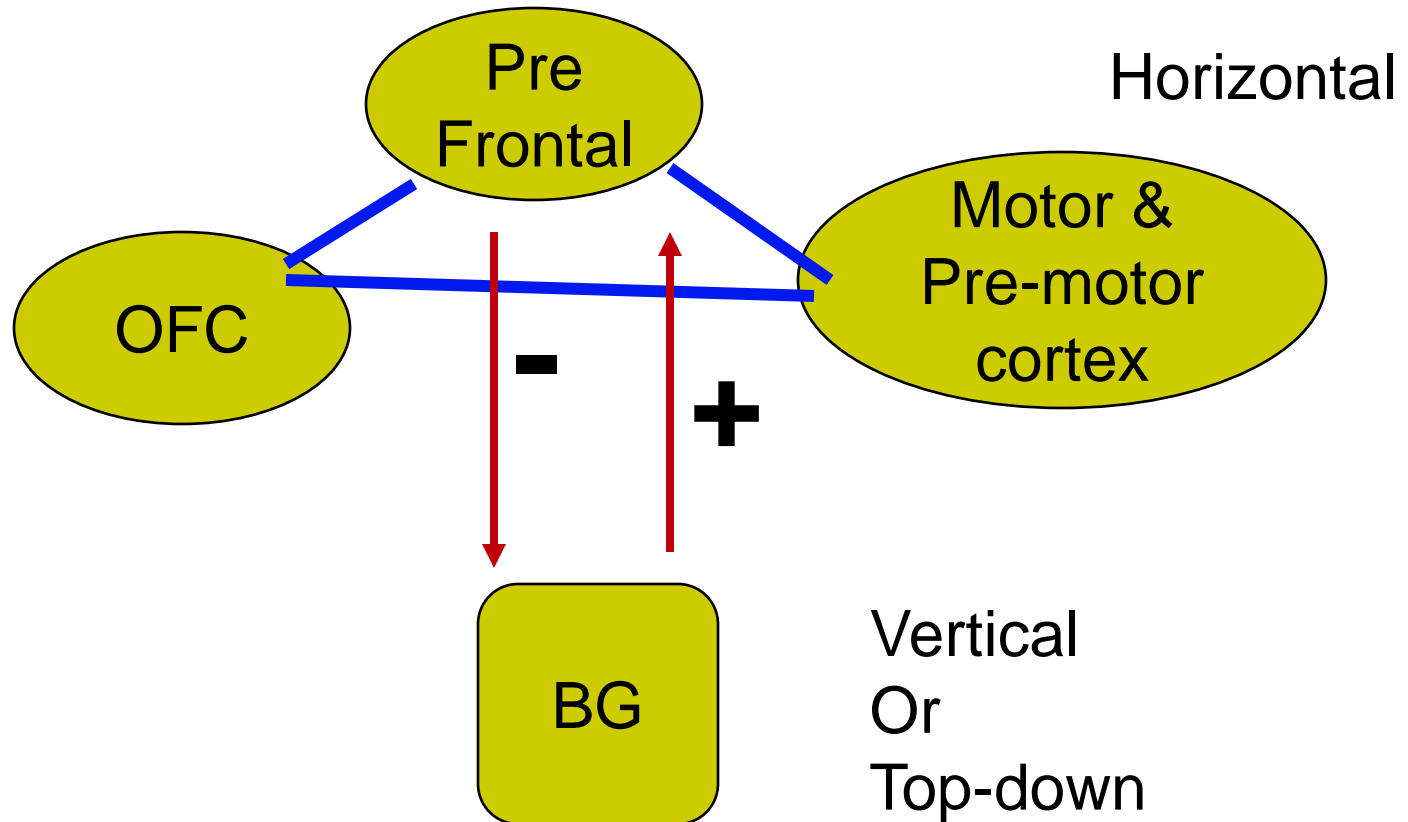
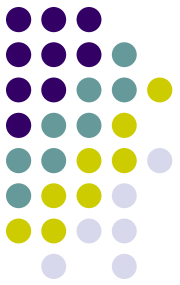
- DA-gic system is involved in **protecting against the emergence of psychotic symptoms**,
- DA-gic system then may stabilize mental homeostatic by spontaneous Down regulation of its own function
- In some patients, this down reg. is sufficient to maintain a non-psychotic state
- if the biological or psychological stresses are so severe that down regulation is not adequate to prevent psychosis.
- The syndrome appears
- **Periodic catatonia: confirmation of linkage to chromosome 15 and further evidence for genetic heterogeneity.**
Periodic catatonia is the first sub-phenotype of schizophrenic psychoses with confirmed linkage despite the existence of considerable genetic heterogeneity.

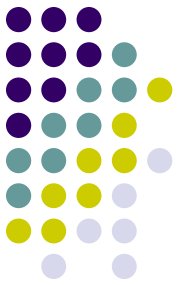
“Top-down modulation”: A Neuropsychiatric hypothesis



- Differential diagnosis of motor symptoms is difficult.
 - Symptoms may have CNS origin [Parkinson's] or psychiatric [catatonia]
 - Despite differences in origin symptoms may appear similar
 - Possibility of dissociation between origin and clinical appearance may reflect functional brain organization in general & cortical-cortical/subcortical in particular.
-
- hypothesized : **similarities and differences between Parkinson's disease and catatonia** - accounted for by distinct kind of modulation between cortico-cortical and cortico-subcortical relation.
 - The different symptoms be accounted for by dysfunction in **orbitofrontal-prefrontal/parietal cortical connectivity reflecting “horizontal modulation” of cortical-cortical relation.**
 - reflecting “vertical modulation” of caudate and other BG by GABA-ergic mediated orbitofrontal cortical deficit may **account for motor symptoms in catatonia.**

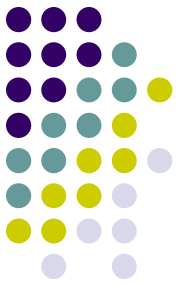
Modulation





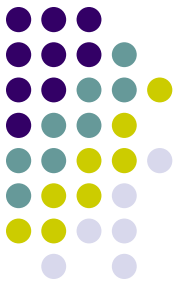
Issues about management

- Over time the mortality has decreased
- Needs emergency and multidisciplinary care
- Often requires Intensive care team
- Management focus has to be on 1. Establishing the diagnosis asap. 2. Arrange supportive treatment, 3. rule out secondary causes. 4. consider definitive treatment, all without loss of time
- Clinical presentation is rarely 'uncomplicated'
- Duration of untreated illness is very high.



Management

- Thus complications of nutritional deficiency,starvations, posturing, metabolic imbalance,infection,worm infestation,skin diseases,injury, cardiac complication,fever and toxemia are common.
- Thus management should address:-
- Vital functions, nutrition, hydration, correction of electrolyte,infection and other relevant condition, and
- Then proceed for definitive treatment with BZ,ECT etc.
- Fitness to anaesthesia and ECT is a crucial clinical issue.



Treatment options

Electroconvulsive therapy and lately benzodiazepines are not only effective treatment options in this form of catatonia, but also helped generate neurobiological hypotheses concerning its pathophysiology

- **LORAZEPAM IV CHALLENGE**

- IF POSITIVE, LORAZEPAM ORAL
 - DOSAGES ARE HIGH- 4-12 MG/DAY

- **ECT**

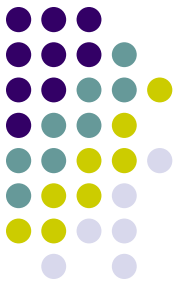
DAILY TREATMENT FOR 2-5 DAYS

BILATERAL ELECTRODE PLACEMENT

- AVOID ANTIPSYCHOTIC DRUGS

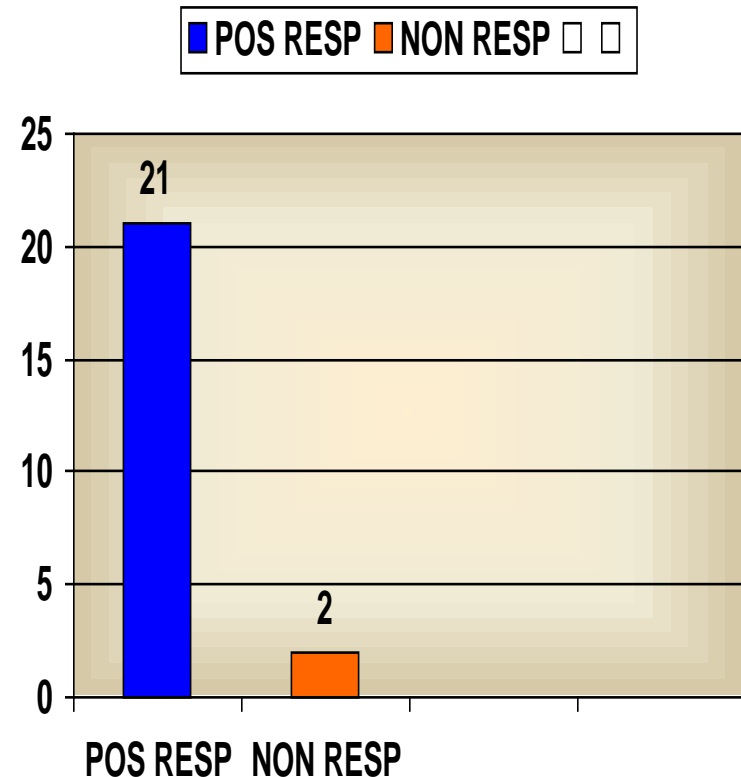
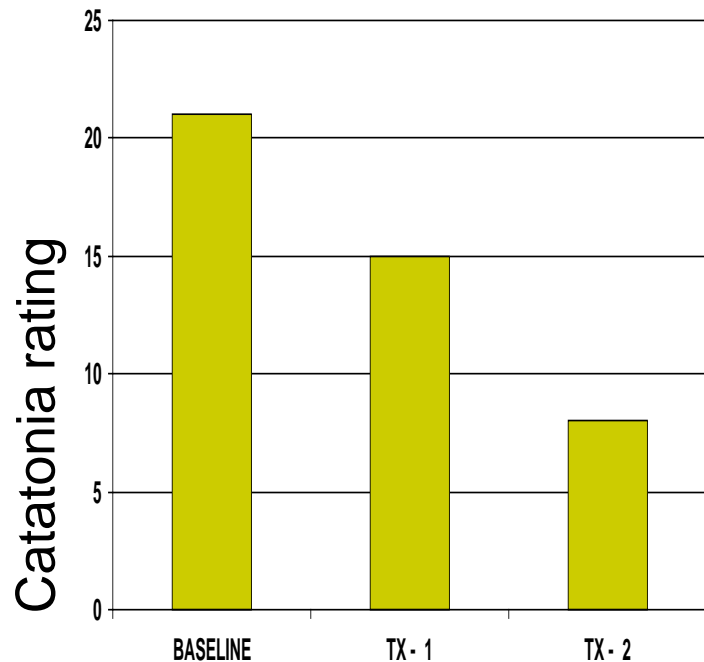
Treatment of catatonia with IV lorazepam

Prospective Study : Bush *et al.*, 1996

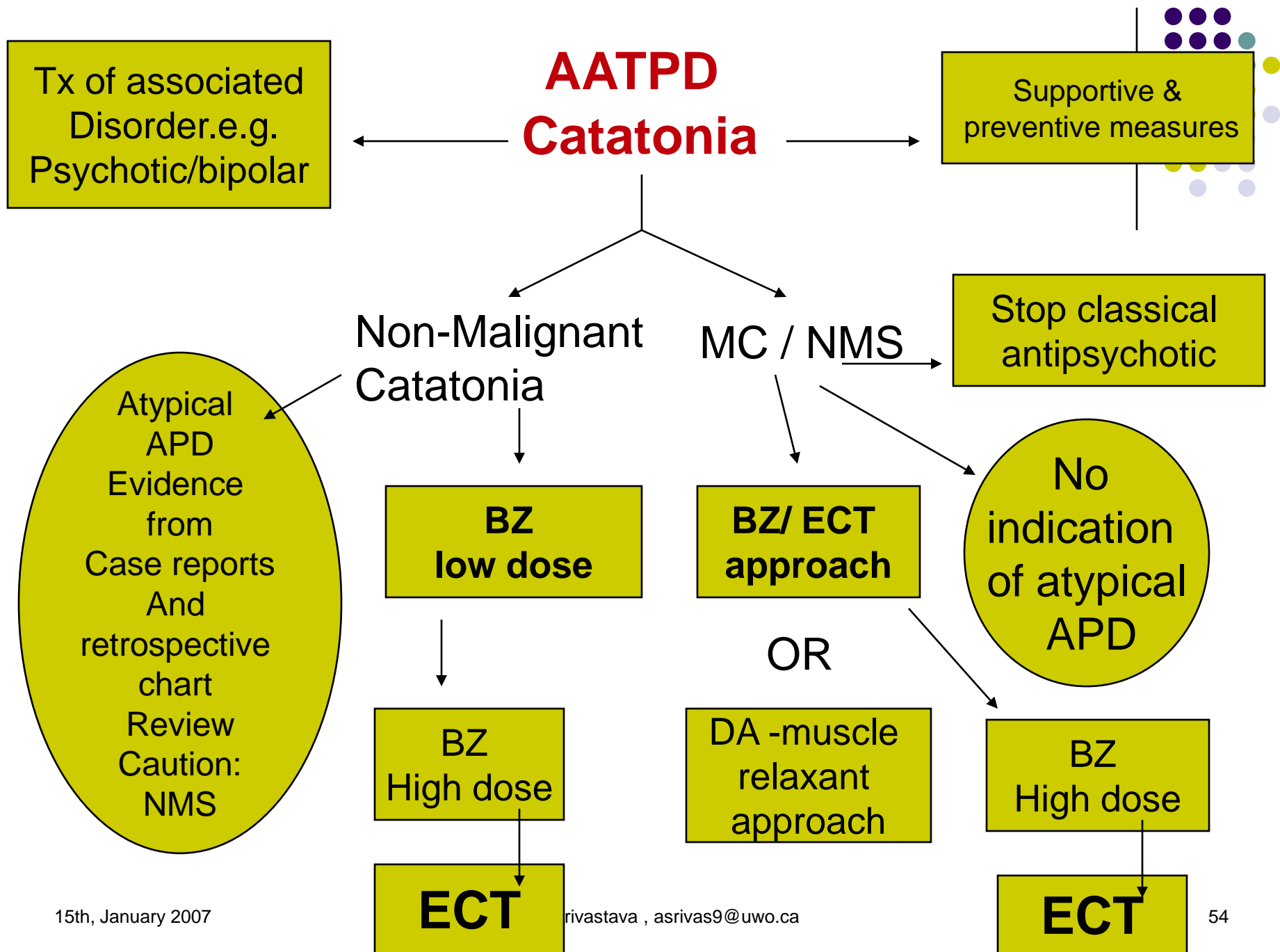


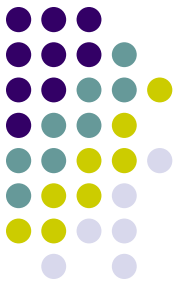
DOSES AT 5 MIN INTERVAL

N=9



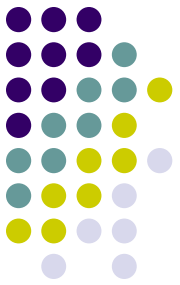
Ungvari : demonstrated that lorazepam was not effective in chronic states of catatonia, associated with chronic psychiatric conditions.-----
 suggesting that not all catatonia are lorazepam responsive





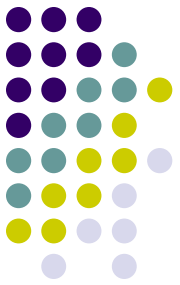
Treatment of Catatonia

- A. simple catatonia(including NL-induced catatonia)
 - ↓
 - Lorazepam
 - Still catatonic
 - DA Agonist
 - Still catatonic
 - ECT
 - B.lethal catatonia (including NMS)
 - Lorazepam
 - And/or
 - DA agonist +/- Dantrolene
 - Still catatonic
 - ECT - prior to 5 days
- Valproate for catatonia: need for caution in patients on SSRIs and antipsychotic.



Advances in management

- **meantime**, may be beneficial in catatonic schizophrenia due to a glutamatergic dysfunction present in catatonic patients.
- **Clozapine** monotherapy for catatonic schizophrenia: should clozapine be the treatment of choice, with catatonia rather than psychosis as the main therapeutic index?
- Clozapine withdrawal catatonia associated with cholinergic and serotonergic rebound hyperactivity: a case report.
- Medical complications of catatonia: a case of catatonia-induced deep venous thrombosis.
- **Gilles de la Tourette** form of catatonia: response to ECT.
- Catatonia and **transcranial magnetic stimulation**.
- Lethal catatonia responding to high-dose olanzapine therapy.
- **Lithium carbonate** in prophylaxis of reappearing catatonic stupor: case report.
- Treatment of catatonic syndrome with **fluoxetine**. Case report]



Advances: future questions

- Nonconscious processing, anterior cingulate and catatonia
- Catatonia is not ready for 'unified theory'
- Does catatonia has a specific brain biology
- What medical catatonia tell us about top down modulation
- Catatonia- a window into the cerebral underpinnings of will
- Catatonia-a disorder of motivation and movement
- Cognitive-motor deficit in catatonia

Finally , its clear that

- 1.our conceptualization of catatonia has changed over years– away from a motor manifestation of schizophrenia and towards a motor,behavioural, emotional manifestation of mixed mania and many other organic disorders
2. its patho-physiology continues to be a mystery.
3. however it is an important clinical syndrome and still continues to be **life threatening condition in neuropsychiatry**

